

AMENDMENTS TO THE CLAIMS

- 1-26. **(Cancelled)**
27. **(Currently amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal exhibiting memory dysfunction and caused by permanent or transient global ischemia, comprising the steps of: determining the existence of memory dysfunction, and administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:
 - (a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and
 - (b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2),thereby reducing memory dysfunction associated with damaged hippocampal tissue in the mammal.
28. **(Previously Presented)** The method of claim 27, wherein said morphogen stimulates synapse formation between hippocampal neurons.
29. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 30-292 of SEQ ID NO:2.
30. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 330-431 of SEQ ID NO:2.
31. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 48-292 of SEQ ID NO:2.
32. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises the amino acid sequence of SEQ ID NO:2.
33. **(Cancelled)**
34. **(Currently Amended)** The method of claim 28, wherein said morphogen comprises a mature form of human OP-1, defined by residues 293-431 of SEQ ID NO: 2.
35. **(Currently Amended)** The method of claim 29, wherein said morphogen comprises a mature form of human OP-1, defined by residues 293-431 of SEQ ID NO: 2.

36. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-2 polypeptide.
37. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-5 polypeptide.
38. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-6 polypeptide.
- 39-42. **(Canceled)**
43. **(Previously presented)** The method of claim 27, wherein the morphogen is administered by intraventricular administration.
44. **(Previously presented)** The method of claim 27, wherein the morphogen is disposed in a biocompatible microsphere.
45. **(Canceled)**
46. **(Currently amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal exhibiting memory dysfunction, comprising the steps of: determining the existence of memory dysfunction, and administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:
 - (a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and
 - (b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2), wherein the damaged hippocampal tissue is damaged by ibotenic acid, ammonia and formaldehyde.
47. **(Canceled)**
48. **(Currently amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal exhibiting memory dysfunction, comprising the steps of: determining the existence of memory dysfunction, and administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:

- (a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2);
and
- (b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2),
wherein the ~~mammal is afflicted with damaged hippocampal tissue is damaged by~~
malnutrition, glucose metabolism disorder, or anorexia.

49-50. **(Canceled)**

- 51. **(Previously presented)** The method of claim 48, wherein the mammal is afflicted with malnutrition.
- 52. **(Previously presented)** The method of claim 48, wherein the mammal is afflicted with a glucose metabolism disorder.
- 53. **(Previously presented)** The method of claim 48, wherein the mammal is afflicted with anorexia.